U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 07 July 2004

CASE NO. 2003-BLA-05404

In the Matter of:

ROSE M. PLEWA,
(widow of JOSEPH PLEWA)
Claimant

v.

BETHENERGY MINES, INC.

Employer

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS,

Party-In-Interest

APPEARANCES:

Robert J. Bilonick, Esq.
For the Claimant

John J. Bagnato, Esq.
For the Employer

Before: Stephen L. Purcell
Administrative Law Judge

DECISION AND ORDER - AWARDING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act of 1977, 30 U.S.C. Section 901 *et seq*.

Benefits under the Act are awarded to coal miners who are totally disabled within the meaning of the Act due to pneumoconiosis, or to the survivors of coal miners who were totally disabled due to pneumoconiosis at the time of their deaths (for claims filed prior to January 1,

1982), or whose death was due to pneumoconiosis. Pneumoconiosis, commonly known as black lung, is a dust disease of the lungs resulting from coal dust inhalation.

The Department of Labor has issued regulations governing the adjudication of claims for benefits arising under the Black Lung Benefits Act at Title 20 of the Code of Federal Regulations. "[T]he procedures to be followed and standards applied in filing, processing, adjudicating, and paying claims" are set forth at 20 C.F.R., Part 725,[1] while the standards for determining whether a coal miner is totally disabled due to pneumoconiosis are set forth at 20 C.F.R., Part 718.

In accordance with the Act and the regulations issued thereunder, the case was referred by the Director, Office of Workers' Compensation Programs, for a formal hearing. The formal hearing was held before the undersigned on October 24, 2003, in Orlando, Florida, at which all parties were afforded full opportunity in accordance with the Rules of Practice and Procedure (29 C.F.R. Part 18) to present evidence and argument as provided in the Act and the regulations issued thereunder, set forth in Title 20, Code of Federal Regulations, Parts 410, 718, 725, and 727. Director's exhibits 1 through 30, Claimant's exhibits 1-10, and Employer's exhibits 1-6 were admitted into the record.[2]

The Claimant is Rose Plewa. She married Mr. Joseph Plewa, the Miner, on November 4, 1978. (Tr. at 19; DX-5). There are no other dependents for purposes of augmentation of benefits. At the formal hearing, Claimant testified that the Miner last worked with Bethlehem Mines. According to Claimant's coal mine work history form, this mine was located in Ebensburg, Pennsylvania. (Tr. at 20; DX-3). Because this claim therefore arises within the territorial jurisdiction of the United States Court of Appeals for the Third Circuit, the precedent of that court applies. *Danko v. Director*, *OWCP*, 846 F.2d 366, 368, 11 BLR 2-157 (6th Cir. 1988). *See Broyles v. Director*, *OWCP*, 143 F.3d 1348, 1349, 21 BLR 2-369 (10th Cir. 1998); *Kopp v. Director, OWCP*, 877 F.2d 307, 12 BLR 2-299 (4th Cir. 1989); *Shupe v. Director*, *OWCP*, 12 BLR 1-200 (1989) (*en banc*).

I have based my analysis on the entire record, including the transcript, exhibits, and representations of the parties, and given consideration to the applicable statutory provisions, regulations, and applicable case law.

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[1] The United States Department of Labor revised the Part 725 regulations effective January 19, 2001. Because this survivor's claim was filed on March 25, 2002, subsequent to the effective date of the amended regulations, the revised regulations apply. *See* 20 C.F.R. § 725.2(c) (2002). *See also* 68 Fed. Reg. 69930-35 (Dec. 15, 2003).

[2] Exhibits submitted by the respective parties are indicated as: "DX" for Director's exhibits, "CX" for Claimant's exhibits, and "EX" for Employer's exhibits. The employer was granted leave to submit the transcripts of depositions of Dr. Bush and Dr. Tomashefski.

Procedural Background and History

The Claimant, Rose M. Plewa, filed a survivor's claim for benefits under the Act on March 25, 2002. (DX-2). On June 5, 2002, the district director issued a Schedule for the Submission of Additional Evidence, and noted that Claimant may be entitled to benefits. (DX-18). The above-named employer did not disagree that it was the proper responsible operator, but contested entitlement and sought to introduce additional evidence. On December 6, 2002, the district director issued a proposed award of benefits. (DX-24). Upon its request for a hearing, this matter was referred on January 31, 2003 to the Office of Administrative Law Judges for a formal hearing. (DX-30).

ISSUE PRESENTED

Whether the Miner's death was due to pneumoconiosis.[3]

FINDINGS OF FACT AND CONCLUSIONS OF LAW

The findings of fact and conclusions of law which follow are based upon my analysis of the entire record, including all documentary evidence admitted, arguments made, and testimony presented.

Factual Background

The miner, Joseph Plewa, was born on March 4, 1932, and died on January 12, 2002. (DX 8). He married his wife, Rose, on November 4, 1978. (DX 5). At the time of his death, the miner was married and living with his wife, who has not remarried since the miner's death. (Tr. at 20). I find that Rose Plewa is the eligible survivor of the deceased miner, and that she has no dependents.

Length of Coal Mine Employment

The Employer originally stipulated to 23 years of coal mine employment. At the hearing, however, the parties stipulated that the miner had twenty-seven years of coal mine employment. (Tr. at 6). I find that this stipulation is supported by the record. I therefore credit Mr. Plewa with twenty-seven years of qualifying coal mine employment.

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[3] Employer has stipulated that this claim is timely, that it is the responsible operator, and that the Miner suffered from coal workers' pneumoconiosis.

Responsible Operator

The employer does not contest its status as the responsible operator. As the record clearly indicates that Bethlehem Mines was the last operator for whom Mr. Plewa worked as a coal miner for a period of at least one year, I find that BethEnergy is properly designated as the responsible operator for this claim.

APPLICABLE STANDARD

The Regulations at 20 C.F.R. Part 718 apply to all claims which are filed on or after April 1, 1980. 20 C.F.R. § 718.1. With respect to survivors' claims, any such claim filed on or after January 1, 1982, is governed by § 718.205(c) of the regulations. Because Mrs. Plewa filed her survivor's claim after January 1, 1982, 20 C.F.R. § 718.205(c) applies to this claim. The regulations provide that a survivor is entitled to benefits only where the miner died due to pneumoconiosis. 20 C.F.R..§ 718.205(a). The Claimant must establish that: (1) the decedent was a coal miner; (2) the decedent suffered from pneumoconiosis at the time of his death; (3) the decedent's pneumoconiosis arose out of his coal mine employment; and (4) the decedent's death was caused by pneumoconiosis or pneumoconiosis was a substantially contributing cause or factor leading to his death. Claimant has the burden of proving each element of entitlement to benefits by a preponderance of the evidence. *Director*, *OWCP v. Greenwich Collieries* [*Ondecko*], 512 U.S. 267, 18 BLR 2A-1 (1994), *aff'g*. *Greenwich Collieries v. Director*, *OWCP*, 990 F.2d 730, 17 BLR 2-64 (3d Cir. 1993). The failure to prove any requisite element precludes a finding of entitlement. *Anderson v. Valley Camp of Utah*, *Inc*., 12 BLR 1-111 (1989); *Perry v. Director*, *OWCP*, 9 BLR 1-1 (1986) (*en banc*).

The survivor of a miner who was totally disabled due to pneumoconiosis at the time of death, but died due to an unrelated cause, is not entitled to benefits. 20 C.F.R. § 718.205(c). If the principal cause of death is a medical condition unrelated to pneumoconiosis, the survivor is not entitled to benefits unless the evidence establishes that pneumoconiosis was a substantially contributing cause of the death. 20 C.F.R. § 718.205(c)(4).

The record contains no evidence of large opacities, massive lesions, or any other condition which a physician has stated could be expected to result in these. There is thus no evidence of record that the Miner suffered from complicated pneumoconiosis. *See* 20 C.F.R. § 718.205(c)(3). I must therefore evaluate the record as a whole to determine whether his death was due to pneumoconiosis, 20 C.F.R. § 718.205(c)(1), or whether pneumoconiosis was at least a "substantially contributing cause" of death. 20 C.F.R. § 718.205(c)(2); *Lukosevicz v. Director, OWCP*, 888 F.2d 1001, 1003, 13 BLR 2-100 (3d Cir. 1989). Pneumoconiosis constitutes a "substantially contributing cause" if it serves to hasten death in any way, however briefly. 20 C.F.R. § 718.205(c)(5). *Soubik v. Director*, *OWCP*, ___ F.3d ___, ___, __ BLR 2-___, No. 03-1668, slip op. at 10 n. 10 (3d Cir. April 30, 2004); *Consolidation Coal Co. v. Kramer*, 305 F.3d 203, 205, 22 BLR 2-469 (3d Cir. 2002) (applying Fourth Circuit law); *Balsavage v. Director*, *OWCP*, 295 F.3d 390, 395, 22 BLR 2-386 (3d Cir. 2002); *Mancia v. Director, OWCP*, 130 F.3d 579, 585, 21 BLR 2-114 (3d Cir. 1997).

The Board has held that, in a Part 718 survivor's claim, the Judge must make a threshold determination as to the existence of pneumoconiosis under 20 C.F.R. § 718.202(a) prior to considering whether the miner's death was due to the disease under § 718.205. *Trumbo v. Reading Anthracite Co.,* 17 BLR 1-85 (1993). The Employer has stipulated that the miner suffered from pneumoconiosis.

In this case, the experts found varying degrees of anthracotic pigmentation. A finding of anthracotic pigmentation, by itself, does not constitute a diagnosis of pneumoconiosis. 20 C.F.R. § 718.202(a)(2) (2002). The autopsy prosector, however, diagnosed anthracosis, Employer's experts do not quarrel with a diagnosis of pneumoconiosis, and Claimant's experts tie the pigmentation to the miner's fibrosis. I find that this stipulation is supported by the evidence, and thus find that Claimant has established the existence of pneumoconiosis.

Medical Evidence

Treatment and Hospital Records

Altoona Hospital

Mr. Plewa was admitted to the Altoona Hospital on December 12, 1993 with complaints of chronic cough of three years duration. The cough, accompanied by shortness of breath, had become worse during the previous two weeks. Dr. G. Sweeney noted hat Mr. Plewa had smoked 30 years before, and that he had been evaluated when he was employed in the coal mines and had been told that he "did not have black lung." Dr. Zlupko examined the miner, and found clear lung fields. This physician had seen Mr. Plewa the previous year, and had thought that his chronic cough was due to chronic sinusitis and nonallergenic rhinitis. A chest x-ray was normal. Mr. Plewa was discharged on December 14 with a diagnosis of "focal bronchitis of the right main stem bronchial tube." (CX-10; DX-10).

Central Florida Regional Hospital

In September, 2000, Mr. Plewa was hospitalized in Sanford, Florida, with indications of prostate cancer. A chest x-ray taken the previous April 14 showed increased interstitial markings. A cardiac catheterization in April detected a moderate coronary atherosclerosis. (DX-10).

Florida Hospital Fish Memorial

The record contains extensive files from this hospital. Mr. Plewa underwent a CT scan, which demonstrated "findings consistent with interstitial lung disease and probable fibrosis." Pulmonary function testing indicated a "moderate restriction." Dr. K. Ariana examined the miner on April 17, 2000 for back problems. The medical history included a reference to "chronic obstructive lung disease," and that condition found its way into Dr. Ariana's list of diagnoses. A pulmonary function study in October, 2000, revealed a "severe restriction."

Mr. Plewa was hospitalized on July 10, 2001, after suffering an acute attack of shortness of breath and cough. Dr. Smith noted after seeing Mr. Plewa that he was "admitted with shortness of breath in the setting of interstitial pulmonary fibrosis." As to history, Dr. Smith noted a "minimal history of tobacco use [and that he] did work in the mines." Mr. Plewa had told the doctor that he had been seen by numerous pulmonologists, and "after undergoing a bunch of x-rays and scan ... was anointed with the diagnosis of idiopathic pulmonary fibrosis." Dr. Smith diagnosed, *inter alia*, coronary artery disease, and also concluded that idiopathic pulmonary fibrosis had been diagnosed in 1993 after "scans, bronchoscopy and multiple medical opinions." He noted as well a "progressive loss of pulmonary function over the last year or so ...." The precipitating factor in the miner's acute shortness of breath was "unclear."

Dr. Skipper's emergency room report noted "acute exacerbation of chronic obstructive pulmonary disease." Dr. Anaya recorded a history of pulmonary fibrosis and hypertension. He recorded no history of obstructive disease. Dr. Anaya found on physical examination diminished breath sounds, no edema or cyanosis. In his July 11, 2001 report, Dr. Anaya diagnosed "acute respiratory distress with hypoxia, idiopathic pulmonary fibrosis as well as prostate cancer, hypertension and coronary artery disease by history." (DX-11).

Dr. Smith conducted an endoscopic bronchial biopsy on July 12. He also noted a history of pulmonary fibrosis and hypertension. Findings from specimens from the bronchial wall of the lower left lung included "anthracotic pigment." A chest CT was cited as showing worsening "interstitial lung disease, especially at the bases."

Mr. Plewa was discharged on July 13 with diagnoses of idiopathic pulmonary fibrosis, hypertension, prostate cancer and coronary artherosclerosis. (DX-11).

Post-Mortem Evidence

Death Certificate

Mr. Plewa died on January 12, 2002. (DX-8). The death certificate listed the immediate causes of death as "respiratory failure," and then "pulmonary fibrosis" as an underlying cause. The cause of death was certified by Dr. Edward Scanlon. (DX-8).

Autopsy

Dr. William Anderson conducted an autopsy on January 13, 2002, with examination limited to the lungs. (DX-9). On gross examination, Dr. Anderson detected "[p]rominent diffuse fibrosis interstitial fibrosis[.]" He also discovered "[d]ense deposits anthracotic pigment with areas of scarring, hyalinization and scattered crystalline material; no atypical changes; areas of mucus plugging of bronchioles with acute inflammation."

Dr. Anderson found:

1. Pulmonary Anthracosis and Diffuse Fibrosis

a. dense aggregates of anthracotic pigment
b. clinical hx: chronic respiratory insufficiency
c. diffuse pulmonary fibrosis and centrilobular emphysema
d. pulmonary edema and moderate congestion
e. areas of hyalinization and scarring in regional nodes with scattered crystalline material consistent with anthraco-silicosis
f areas of bronchial mucous plugging with acute peribronchial inflammation
[g.] no diffuse pulmonary acute inflammatory changes

He further observed that the "[d]ense deposits anthracotic pigment are identified diffusely within the pulmonary parenchyma and in the lymph nodes of the perihilar region." Dr. Anderson opined that the cause of death was "pulmonary anthracosis and diffuse fibrosis consistent with anthraco-silicosis: clinical 'black-lung disease[.]'" Employer's counsel represented at the hearing that Dr. Anderson is board certified in both clinical and anatomic pathology. (Tr. at 14). This representation is corroborated by the doctor's CV, which shows board certification in anatomic, forensic, and clinical pathology. Dr. Anderson has also been a clinical instructor at the Emory University School of Medicine, and Clinical Assistant Professor, SUNY, Stony Brook, in New York, in 1987-88.

Medical Reports

Dr. Stephen T. Bush

Dr. Bush conducted a review of the miner's medical records, examined the pathology materials, and issued his report on August 15, 2002. (DX-12). He noted that the miner had been hospitalized with various complaints over the years. Based on his review of the records to date, Dr. Bush concluded that the miner suffered from a "minimal degree of simple coal worker's pneumoconiosis." He wrote that "[l]ess than 1 percent of the lung tissue is affected by these lesions, indicating a very mild degree of coal worker's pneumoconiosis." Dr. Bush also diagnosed "idiopathic pulmonary fibrosis," and opined that "Mr. Plewa died of respiratory failure due to idiopathic pulmonary fibrosis causing end-stage lung disease." He continued that the "very mild degree of simple coal worker's pneumoconiosis played no role in death due to the very limited degree and extent of this disease." He also stated that pneumoconiosis and coal mine dust exposure played no role in the development of the miner's idiopathic pulmonary fibrosis. Dr. Bush explained:

> The radiologic findings indicated in interstitial changes are most consistent with idiopathic pulmonary fibrosis rather than the fibronodular lesions of coal worker's pneumoconiosis. These findings were confirmed on chest CT examination. The pulmonary function studies showed severe restrictive disease without the obstructive component found in coal worker's pneumoconiosis. The restrictive disease is typical of idiopathic pulmonary fibrosis.

> The progressive nature of the fibrotic process in the lungs is typical of idiopathic pulmonary fibrosis. The course of this disease varies considerably. Mr. Plewa died six (6) years after the diagnosis of idiopathic pulmonary fibrosis was made by Dr. Stauffer (03/25/96). This survival period is consistent with published reports of a median survival for patients of up to six (6) years[.]

Dr. Bush then concluded that Mr. Plewa "would have died at the same time and in the same manner of idiopathic pulmonary fibrosis if he had never been exposed to the pulmonary hazards of coal mining employment." The doctor is board certified in anatomic and clinical pathology, with a "special competence" in medical microbiology. He has practiced in Johnstown, the coal mining region. He has served as a Clinical Assistant Professor, Department of Pathology at the University of Alabama Medical Center. (DX-12). Dr. Bush has not been utilized by the Department of Labor as a pathologist in regard to the Black Lung program. (EX-4 at 16). Since 1969, Dr. Bush, who retired from full-time practice in 1998, has conducted between five and ten autopsies on decedent miners per year. (EX-4 at 17). He has concluded that in "perhaps one percent of the cases" simple coal workers' pneumoconiosis was the cause of death in approximately 500 cases reviewed by him as a consultant. (EX-4 at 18-20).

Dr. Joshua A. Perper

Claimant introduced a medical report, submitted on September 20, 2002, by Dr. Joshua A. Perper. (DX-13). Dr. Perper reviewed the miner's medical records, including the death certificate, autopsy protocol and slides, as well as hospitalization records. He also reviewed the medical report from Dr. Bush.

Dr. Perper noted that physicians in 1992 recorded coal mine work histories of from 30 to 33 years. Mr. Plewa also smoked for 12 years at the rate of one pack per week. Turning to Dr. Bush's report, Dr. Perper noted that Dr. Bush did not discuss the miner's occupational history, and did not "mention the autopsy findings or diagnoses[.]" In a lengthy critique, he took issue with the thoroughness of Dr. Bush's report and its final conclusions. Dr. Perper concluded his discussion of Dr. Bush's report by noting:

> [I]t is unreasonable to label a process of pulmonary fibrosis as idiopathic in general when there is clear evidence of exposure to a fibrotic agent such as mixed coal containing silica, both by occupational history and through presence of significant anthraco-silicosis due to coal workers' pneumoconiosis. This is particularly so in the case of Mr. Plewa in view of his significant coal workers' pneumoconiosis. ...[T]he report of Dr. Bush is inadequate in scope of reported findings and inaccurately downgraded and misinterpreted the significance of the pneumoconiotic process.

After examining the seven autopsy slides of record, Dr. Perper detected "[p]leural and sub-pleural fibro-anthracosis with presence of birefringent silica crystals." H also saw in a few places "small calcified and ossified foci" present in the areas of fibro-anthracosis. Also noted

were numerous desquamated cells in the bronchi, and in the lung tissue slides he observed "areas of severe interstitial fibrosis with anthracotic pigmentation," some chronic emphysema and "numerous macrophages loaded with anthracotic pigment." Dr. Perper reported that "even in places where the pulmonary architecture is fairly well preserved there are anthracotic macules around blood vessels, airways and in the inter-alveolar septa." This examination also revealed micronodules scattered through the pulmonary paranchyma and signs of focal emphysema. Dr. Perper pointed to figures, present in this record as color images of slides, that demonstrated "an aggregate of anthracotic nodules surrounded by less dense fibro-anthracotic tissue."

In the final analysis, Dr. Perper detected numerous examples of anthracosis and anthracotic tissue. He said that "a careful examination of all the lung sections failed to disclose any granulomas, other than the pneumoconiotic nodules." Dr. Perper diagnosed "coal workers' pneumoconiosis, interstitial type on background of macular and micronodular coal dust type and silicotic type pneumoconiosis, severe[;]" focal emphysema and sclerosis of intra-pulmonary blood vessels consistent with pulmonary hypertension.

The doctor concluded that the "degree and severity of the pulmonary process of Mr. Plewa's coal workers' pneumoconiosis, as substantiated by the pulmonary autopsy findings was extensive and severe, and associated with severe clinical symptomatology." He opined that the miner's "severe coal workers pneumoconiosis was the primary cause of his death, a substantial cause of his death and definitely hastened his death."

Dr. Perper is board-certified in anatomic, forensic, and surgical pathology. He was an Associate Medical Examiner in Bethesda, Maryland from 1967 until 1972, the Chief Forensic Pathologist for the Allegheny County, Pennsylvania Coroner from 1972 until 1980, from the latter date until 1994 was the Coroner of Allegheny County, and has been the Chief Medical Examiner of Broward County, Florida, since 1994. He has served as Clinical Professor of Pathology at the University of Pittsburgh and the University of Miami. In addition to teaching in the field of pathology, Dr. Perper has instructed in the area of epidemiology. (CX-6). He has performed several hundred autopsies on decedent miners to assess the cause of death. (CX-6 at 5).

Dr. Stephen T. Bush

On October 17, 2002, Dr. Bush submitted a rebuttal to Dr. Perper's report. (DX-14). He differed in some respects with Dr. Perper's theories, and faulted Dr. Perper's use of photomicrographs that were too magnified.[4] Stating that "[o]nly a thorough microscopic examination of the histologic slides correlated with the gross pathology, lifetime radiographic findings, pulmonary assessments and clinical evaluations can lead to a diagnosis with reasonable degree of medical certainty." Acknowledging that pneumoconiosis can progress after the cessation of exposure, Dr. Bush nevertheless insisted that Mr. Plewa's pneumoconiosis was "too limited in degree and extent to have contributed to pulmonary failure and death."

[4] Dr. Perper responded to Dr. Bush's criticism of the use of the microphotographs. He testified in his deposition that they are illustrative of the findings rendered by him, and the findings on gross examination. (CX-6 at 30).

Dr. Joseph F. Tomashefski

Dr. Tomashefski is Chairman of the Department of Pathology, MetroHealth Medical Center, and full Professor of Pathology at the Case Western Reserve University. He is board certified in anatomic and clinical pathology, and is widely published in the field. (EX-1). Dr. Tomashefski does not conduct consulting evaluations for the Department of Labor Black Lung program. (EX-6 at 17). He testified that he has not performed an autopsy on a decedent miner "for several years[,]" and estimated that he had performed about five or six autopsies on decedent miners over the last ten years. (EX-6 at 18). Dr. Tomashefski could not recall a case in the two years preceding the deposition where he has opined that coal workers' pneumoconiosis played a significant and substantial role in the cause of death. (EX-6 at 22-23). Nevertheless, Dr. Tomashefski emphasized that being conversant in pulmonary pathology places him in a better position to assess occupational disorders of the lung, and pointed out as well that forensic pathologists have consulted him with respect to occupational diseases. (EX-6 at 30).

He was asked by Employer to review the medical file and pathology materials, and on October 30, 2002, issued a detailed and comprehensive report of his findings. (EX-1).

Upon reviewing the autopsy slides, Dr. Tomashefski detected, *inter alia*, "extensive, diffuse, interstitial fibrosis with honeycomb change." He found a "mild to moderate amount of black pigment and birefringent crystals ... concentrated in the subpleural area and focally around blood vessels." Dr. Tomashefski continued that "only a small amount of pigment and/or crystalline material is associated with diffuse fibrosis." The amount of coal macules and micronodules found in the lung parenchyma was "rare," and their size was "small (<1 mm diameter)."

Dr. Tomashefski diagnosed "advanced diffuse interstitial fibrosis, consistent with idiopathic pulmonary fibrosis (IPF)." He opined that the miner's death was respiratory failure due to IPF. The miner also suffered from a mild simple coal workers' pneumoconiosis, but Dr. Tomashefski explained that the pulmonary fibrosis was associated with the idiopathic pulmonary fibrosis and not due to the effects of coal mine dust exposure. He explained that the "distribution and pattern of fibrosis is typical of that associated with IPF[,]" and concluded:

> The degree of simple coal workers' pneumoconiosis which is documented in Mr. Plewa's lung tissue, in my opinion, is too mild to have caused him any respiratory symptoms or exercise limitation. It is also too mild to have caused, or been a contributory factor in, Mr. Plewa's death.
>
> Within reasonable medical certainty, Mr. Plewa would have died at approximately the same time and in the same manner even if he had never worked as a coal miner or developed mild simple coal workers' pneumoconiosis.

Dr. Waheeb Rizkalla

Dr. Waheeb Rizkalla reviewed the miner's medical records and submitted a medical report on November 19, 2002. (CX-1). He is a board certified anatomical and clinical pathologist, and has conducted approximately 400-500 autopsies on decedent coal miners. (CX-8 at 3-5). He opined that the miner was afflicted with coal workers' pneumoconiosis, and said that this disease was a "substantial contributing factor in his death." He disagreed with the conclusions rendered by Drs. Bush and Tomashefski, who opined that the miner had suffered from idiopathic pulmonary disease. He explained that he had observed that the silica detected by his examination "is a fibrogenic element," and posited that the interstitial pulmonary fibrosis was "secondary to his occupational disease and not idiopathic pulmonary fibrosis."

Dr. Rizkalla wrote that the "usual and typical forms of coal worker's pneumoconiosis are macules, micronodules or macronodules." He detected "micronodules ranging up to 2.0 mm to 3.0 mm." The doctor concluded that the "overwhelming response" to the miner's coal dust exposure is "interstitial pulmonary fibrosis."

Dr. Rizkalla, quoting from a peer-reviewed article, wrote that "[d]iffuse interstitial pulmonary fibrosis has been identified in autopsies of coal miners in some studies." He opined as well that "[c]onsidering the pulmonary fibrosis ... as idiopathic pulmonary fibrosis type despite his occupational history as a coal miner with exposure to coal dust is unjustified."

Dr. Rizkalla concluded:

> Mr. Plewa died from respiratory failure secondary to his interstitial pulmonary fibrosis which was induced by coal dust exposure. He suffered from hypoxia which required oxygen therapy even prior to his death. I am in disagreement with the pathologists who consider his interstitial pulmonary fibrosis of idiopathic type. I consider it secondary to coal dust exposure and his coal worker's pneumoconiosis as a substantial contributing cause of his respiratory failure and ultimate death.

Dr. Joseph F. Tomashefski

Dr. Tomashefski submitted a supplemental report in rebuttal to Dr. Rizkalla's report on April 22, 2003. (EX-2). He disagreed with Dr. Rizkalla's conclusion that the miner's interstitial pulmonary fibrosis was caused by coal mine dust exposure, and reiterated his view that this condition represents idiopathic pulmonary fibrosis. Dr. Tomashefski explained that the clinical course, symptoms, physical findings and radiographic features of Mr. Plewa's pulmonary condition were "typical" with IPF. He cited the miner's "bibasilar crackles, and the radiographic distribution of peripheral and lower lobe interstitial fibrosis[,]" features not seen in coal workers' pneumoconiosis. Dr. Tomashefski also explained that the "pattern of interstitial fibrosis and lung remodeling ... is typical of that seen in IPF."

To buttress his conclusion that Mr. Plewa's diffuse interstitial fibrosis is unrelated to coal

mine dust exposure or to coal workers' pneumoconiosis, Dr. Tomashefski noted the "minimal evidence of deposition of mineral dust or coal dust, and no reaction to either silica or coal dust in the areas of diffuse interstitial fibrosis due to silica." He observed only very mild simple coal workers' pneumoconiosis in areas that were outside of the areas of diffuse interstitial fibrosis.

Dr. Tomashefski faulted the theoretical underpinning for Dr. Rizkalla's conclusions, stating his view that the treatises cited by that pathologist did not support Dr. Rizkalla's opinion as to the etiology of Mr. Plewa's interstitial fibrosis.

Dr. John T. Schaaf

Dr. Schaaf was asked to review the miner's medical file, and submitted a consultation report on May 8, 2003. (CX-3). He affirmed that Mr. Plewa "acquired coal workers' pneumoconiosis during his life," citing the pulmonary fibrosis detected on chest x-ray and that such a diagnosis was "confirmed histopathologically." He also opined that coal workers' pneumoconiosis was a substantial contributor or that it accelerated the miner's death.

The doctor reasoned that Mr. Plewa's death was due to a progressive pulmonary dysfunction, and that the miner's fatal pulmonary fibrosis was in his opinion "synonymous with his coal workers' pneumoconiosis." To him it was "clear" that Mr. Plewa's coal mine employment "led to a disease that caused his death." Dr. Schaaf explained:

> This disease was described clinically as pulmonary fibrosis and pathologically has been confirmed to be fibrosis and pneumoconiosis. This clinical, radiographic and histologic picture is consistent with the medical literature which demonstrates an excess incidence of a predominately fibrotic as opposed to nodular type of pneumoconiosis associated with coal mine employment.

Dr. Schaaf submitted a follow-up letter report on July 2, 2003, in response to Dr. Tomashefski's report. (CX-4). Dr. Schaaf was unconvinced by Dr. Tomashefski's contrary opinions. He explained that "Mr. Plewa has an environmental exposure which is known to produce fibrosis." Dr. Schaaf also pointed out that Dr. Tomashefski's diagnosis of IPF "implies that we have no underlying etiology and is therefore a diagnosis of exclusion."[5] Given the miner's coal mine employment, "there is evident and known etiology for his pulmonary fibrosis; i.e. coal mine employment and coal dust exposure[,]" the doctor concluded.

Dr. Schaaf is board-certified in internal medicine with a subspecialty in pulmonary disease, and is likewise board-certified in critical care medicine (CX-5).

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[5] Dr. Perper was questioned about Dr. Schaaf's use of this description of IPF as a "diagnosis of exclusion." He said that to make such a diagnosis, one would have to exclude all other potential causes. (CX-6 at 42).

Dr. Stephen T. Bush

Dr. Bush testified at a deposition taken on August 15, 2003 (EX-4). He opined that Mr. Plewa died as a result of "chronic obstruction — as a result of pulmonary fibrosis, which led to respiratory failure." (EX-4 at 21). He attributed this terminal condition to idiopathic pulmonary fibrosis. (*Id*. at 22). Dr. Bush explained that the miner displayed many of the signs and symptoms of that disease process, and the diagnosis of IPF was likewise confirmed by his analysis of the autopsy slides.

Dr. Bush cited the presence of bibasilar crackles on physical examinations, a characteristic sound from lower lung fields, in support of his view that IPF was the interstitial fibrotic disease at work in this case. Patients with IPF, he said, have a "lower lobe involvement" such as that exhibited by Mr. Plewa. These patients, and Mr. Plewa, also typically have a restrictive lung disease as opposed to an obstructive impairment. (*Id*. at 23-24). This disease process was shown by the interstitial fibrosis of the lung. The miner's coal workers' pneumoconiosis, while present, was to Dr. Bush "very limited and consisted of small micro-nodules ... with a localized fibrous reaction ... and had a minimal amount of focal dust emphysema." (*Id*. at 25-26). Dr. Bush thought that less than one percent of the lung was affected by coal workers' disease.

The doctor could not relate pneumoconiosis to the miner's death. He noted that scientific literature indeed describes instances where coal mine dust produces diffuse fibrosis, but stated that the fibrosis in those cases is accompanied by a "large quantity of dust pigment ... which Mr. Plewa did not exhibit." Dr. Bush thought that the pigment which was present was random. He was emphatic that, had Mr. Plewa not worked in the mines, he would have died at the same time and in the same manner in the absence of coal mine dust exposure. Dr. Bush thought this clinical course typical of patients he has examined with IPF. (*Id*. at 27-28)

Dr. Bush explained his disagreement with the conflicting opinions by Drs. Perper and Rizkalla. Those experts, he explained, diagnosed what to Dr. Bush is an "unusual reaction to coal dust" with the "typical coal worker lesions[.]" Dr. Bush would require seeing a greater amount of dust in the lungs before he would render an opinion that pneumoconiosis was involved in the development of Mr. Plewa's fatal disease.

On cross-examination, Dr. Bush acknowledged that he did not diagnose emphysema, save for a reference to focal emphysema in his August, 2002, report. He explained that he did not dwell on the miner's coal mine exposure history because he assumed that there was sufficient exposure to cause the miner's pneumoconiosis. Nor did he originally discuss the findings of the autopsy prosector. (EX-4 at 35). Dr. Bush noted that the prosector's use of the phrase "consistent with," in referring to the autopsy findings "consistent with anthracosilicosis," is qualified or uncertain. Dr. Bush also explained that "clinical Black Lung just refers to the fact that he had pulmonary symptoms. I don't know of anybody who made a diagnosis of Black Lung Disease." (*Id*. at 36). Although both Drs. Perper and Rizkalla had noted emphysema, Dr. Bush had not written about it in his report. (EX-4 at 42-3). When questioned about the prosector's findings of emphysema, Dr. Bush explained that to him it was "a localized process and I did not dwell on it." Although he noted the disease, he did not address it in his written

report because it was considered too trivial. (EX-4 at 44).

Dr. Bush believed that the emphysema present here would have been "multi-factorial" in origin. When asked whether coal dust exposure would be one of the factors, the doctor explained that coal mine dust exposure causes a focal emphysema, and that central lobular emphysema is more likely the effect of the diffuse interstitial fibrosis, the miner's smoking history or perhaps episodes of bronchitis. (*Id*. at 47). The doctor discounted the miner's coal mine employment exposure, because the emphysema, of any type, when compared to the lung disease that caused the miner's problems, is a "trivial finding," as is the pneumoconiosis that was also diagnosed. (EX-4 at 49).

The deposition was resumed on November 14, 2003. (EX-5). When questioned extensively about the duration of the miner's complaints of cough and shortness of breath, which apparently became manifest at a point earlier than was reflected in his reports, Dr. Bush acknowledged that respiratory complaints of this nature were of a longer duration. (*Id*. at 24-25). He continued to assert that Dr. Perper's criticism, that he underestimated the duration of the miner's respiratory complain history, was wrong. (EX-5 at 29). When asked whether coal workers' pneumoconiosis causes a restrictive or obstructive lung disease, Dr. Bush replied that "this is better answered by a pulmonologist than a pathologist, but my understanding is we typically see a mixture of obstructive and restrictive findings." (*Id*. at 31).

On redirect examination, Dr. Bush was questioned about a medical treatise by Drs. Churg and Green, and in particular their treatment of the issue of coal workers' pneumoconiosis. He stated that the miner's clinical course differed significantly from the "relatively benign" course outlined in the treatise of patients afflicted with interstitial disease due to coal mine dust exposure. (*Id*. at 34). He explained that his opinion, that Mr. Plewa's interstitial fibrosis was not due to coal mine dust exposure, was based primarily on the slides, "[a]nd primarily the finding of diffuse fibrosis in the lungs, not associated with dust or coal worker lesions." (*Id*. at 35-36). Dr. Bush did see areas of dust pigment, but did not associate it with the fibrotic process, which involved both pigmented and non-pigmented areas. (*Id*.) He also acknowledged that where the fibrosis is pigmented with diffuse dust pigment, that he would be "willing to accept that this is due to coal mine dust exposure." But the doctor would not concede this association where there is diffuse fibrosis and only some pigment. (*Id*. at 38).

On further cross-examination, Dr. Bush agreed that the idiopathic pulmonary fibrosis was more than enough to have resulted in the miner's death. He also agreed that, based on his notes, half of this process was involved with pigmentation. (*Id*. at 49). Moreover, he conceded that the only "fibrogenic exposures that were identified ... at the time of [the miner's] death were caused by ... his coal dust exposure." (*Id*. at 50).

Dr. John T. Schaaf

Dr. Schaaf was deposed on September 26, 2003. (CX-7). Claimant secured this testimony based on a review of the miner's medical records. He agreed with other medical experts of record that Mr. Plewa died a "respiratory death." (*Id*. at 17). Dr. Schaaf testified that the symptoms of idiopathic pulmonary fibrosis and coal workers' pneumoconiosis are "probably

very much the same." He opined, however, that the miner did not die from the former, but from pneumoconiosis. He cited the miner's coal workers' pneumoconiosis, an identifiable disease process that constituted a known cause. Dr. Schaaf stated that, as a result, "we should not be invoking the term idiopathic." (*Id*. at 19). He noted documentation in professional literature that diffuse interstitial fibrosis is associated with coal workers' pneumoconiosis, and that it would be reasonable to ascribe the miner's pathology to pneumoconiosis.[6] He reiterated that pneumoconiosis is a "fibrogenic exposure," and that the miner suffered no other such exposure of consequence. (*Id*. at 25). Indeed, Dr. Schaaf pointed out that the birefringent crystals and the coal dust deposits were part of coal workers' pneumoconiosis.

Dr. Schaaf observed that the autopsy prosector's finding of a "pattern of diffused centrilobular emphysema" indicated that emphysema was present in the entire lung, that "it's all over the lung." He also noted Dr. Anderson's observation of "dense deposits of anthracotic pigment," and stated that the reference to diffuse in this case meant its presence in the entire lung. He agreed that, based on the gross description, there was sufficient evidence of dust deposition and pigmentation to cause a fibrotic interstitial reaction. He also agreed that the prosector's findings as to the severity of the "dust pigmentation and coal dust burden on the decedent's lungs ... as well as the findings of coal worker's pneumoconiosis," were confirmed by the reports and opinions of Drs. Perper and Rizkalla.

The doctor concluded that the miner died of coal workers' pneumoconiosis, and that it "hastened his death and significantly contributed to his death." He disagreed with Dr. Bush that Mr. Plewa suffered from idiopathic pulmonary fibrosis. Dr. Schaaf also emphasized that neither the opinions of Dr. Bush nor the contrary conclusions by Dr. Tomashefski would cause him to change his conclusions regarding the role played by pneumoconiosis in this case. (*Id*. at 42). He further posited that, where an abundant amount of coal dust had been identified, it would be reasonable to relate that exposure as the cause of the miner's interstitial fibrosis.

On cross-examination, Dr. Schaaf explained why an idiopathic disease would not be appropriate where there is a cause:

> [I]f a patient has a disease that is interstitial disease and has a lot of fibrosis then I am obligated as a physician to look for a cause, and amongst the causes will include fibrogenic dust exposure.
>
> If I find a cause for the disease process then the label or the diagnosis is whatever that disease is, and it's not idiopathic.

(CX-7 at 47). On redirect, Dr. Schaaf testified that the "gold standard" for the diagnosis of certain disease processes would be findings by pathologists. (CX-7 at 52-53).

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[6] Dr. Schaaf referenced an article in the Atlas of Nontumor Pathology, published by the Armed Forces Institute of Pathology. (CX-7, Deposition Exhibit 2).

Dr. Joshua A. Perper

Dr. Perper's deposition testimony was recorded on October 6, 2003. (CX-6). He recounted that the gross findings from the autopsy and the slides were consistent.[7] He was impressed first by the fact that the lungs were "heavy," and this heaviness was explained by the nature of the pathology found. (*Id*. at 13). Dr. Perper was impressed by the prosector's findings of "dense deposits of anthracotic pigment diffusely throughout the pulmonary parenchyma and in the lymph nodes of the hilar area ... in a context of a fibrotic lung is a very significant finding[.]" He explained that the prosector's findings "document the presence of severe fibrosis, interstitial fibrosis of lung with marked anthracotic pigmentation and significant associated centrilobular emphysema with bullae and corresponding heavy lungs." (CX-6 at 14). The doctor's microscopic examination is consistent with the gross findings.

Dr. Perper agreed with the conclusion reached by the autopsy prosector that the miner's death was caused by "black lung." He disagreed with Dr. Bush's description of the term "consistent" as ambiguous, and stated that the use of this word does not entail ambiguity in a diagnosis, and indeed constitutes a firm diagnosis. He saw no uncertainty in Dr. Anderson's diagnosis and attribution of cause of death. (*Id*. at 18-19).

The doctor noted the importance of findings with respect to the peri-bronchial lymph nodes, explaining that while these lymph nodes would not show pulmonary damage, they are indicative of the severity of the exposure.[8] (*Id*. at 24). He further concluded that "this type of interstitial fibroanthracosis is clearly related to the presence of coal dust containing silica because the — the severity of the process is consistent with that." The doctor was emphatic in his findings:

> This is not a condition in which the anthracosis is minimal. This is a situation in which the interstitial fibrosis and anthracosis are closely married together and they have the presence of the silica crystal. ... In this particular case this is not a borderline finding. This is not a slight finding. This is not a remote finding.

(CX-6 at 27).

Dr. Perper was asked about studies that he had cited, and whether, in his opinion as a professor of epidemiology, those studies and references he has used are useful in understanding

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[7] Dr. Perper emphasized that a review of gross findings was an important complement to a histological review, because it provided the context for an examination of the microscopic slides. (CX-6 at 12).

[8] "[T]heir almost complete replacement by silicotic nodules is a marker that this was a severe exposure to coal dust which is consistent with the lung's occupational exposure of more than 30 years and it's also congruent with the severe coal workers' pneumoconiosis of the interstitial and micro nodular type and silicotic type evident in the lung tissue." (CX-6 at 24).

the disease processes involved. He agreed that this information would be of assistance.[9] (CX-6 at 35-36).

He opined that the interstitial fibrosis that was present was "diffuse," and affected the totality of the lungs. He disagreed with Dr. Bush's assertion that half of the fibrosis is associated with pigmentation, and half was not. (*Id*. at 37-38). He agreed with Dr. Schaaf's opinion that the effects of coal dust and silica inhalation can go "beyond the areas readily identified as anthracotic." (*Id*. at 38). The silica, he said, would not appear as anthracotic but as crystals or birefringent silica.

Dr. Perper clearly disagrees with the diagnosis of IPF, called by Dr. Schaaf as a diagnosis of exclusion, because here there "was a fibrogenic agent which is universally recognized in the tissue, in the lung tissue with the corresponding long term exposure explaining this accumulation."

Dr. Perper voiced his concurrence with the opinions of Drs. Schaaf and Rizkalla, and his continuing disagreement with the conclusions reached by Drs. Bush and Tomashefski. He described the mechanics of Mr. Plewa's death as:

> [P]ulmonary impairment and failure resulting from the involvement of the lung breathing tissue by severe interstitial type of coal workers' pneumoconiosis on the background of macular and micro nodular coal workers' pneumoconiosis.

(CX-6 at 44-45). He disagrees with Dr. Tomashefski's suggestion that there was not an abundant presence of mineral dust in the areas of fibrosis. Instead, Dr. Perper concluded, there was an abundant presence of mineral dust in the areas of fibrosis, a finding that correlated that finding made by the autopsy prosector. (*Id*. at 47). He also disagreed with Dr. Tomashefski's conclusion that the "diffuse interstitial fibrosis is largely non-pigmented[.]" Instead, Dr. Perper reiterated that "the interstitial fibrosis contains significant and advanced areas of anthracotic pigmentation [and that] all areas of the lung you have either anthracotic pigmentation or silica crystals or both indicating the cause of the anthracofibrosis." He pointed out that even Dr. Bush found a greater amount of pigmentation than did Dr. Tomashefski. (*Id*. at 49).

Dr. Perper identified two exposures in this case. The first was the miner's smoking, which was not considered substantial, and coal dust and coal workers' pneumoconiosis.

Dr. Waheeb M. Rizkalla

Claimant obtained the deposition testimony of Dr. Rizkalla on October 22, 2003. (CX-8). The doctor was questioned about his review of the miner's medical records and the other medical reports in this claim. He agreed with the other experts who have rendered opinions that the miner died a "respiratory death." (*Id*. at 10-11).

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[9] Dr. Tomashefski is of the view that being a forensic pathologist or epidemiologist would not assist in an assessment of an individual case. (EX-6 at 30-31).

Dr. Rizkalla defined interstitial pulmonary fibrosis as a condition "that will be secondary to exposure to coal dust or substantial exposure to coal dust with the carbon and silica particles initiating the fibrotic process." (*Id*. at 11). He then defined idiopathic pulmonary fibrosis as:

> [A] fibrosis of the lungs for unknown reason and that occurs usually on a healthy person without previous exposure to an occupational dust or hazardous material or any material that is known to be fibrogenic to the lung, and all of a sudden the patient will present with a respiratory ailment and respiratory dysfunction. In the absence of any known etiology, we use the term. Idiopathic means that we don't know the cause and [there is] no apparent cause to induce death.

(CX-8 at 12).

In this case, Dr. Rizkalla opined that the "fibrogenic factor in the lungs was the silica and carbon particles[;]" no other fibrogenic agents were identified. (*Id*.). Dr. Rizkalla characterized the degree of pneumoconiosis in this miner as "moderately severe." He explained that "the deposition of the particles was identified in every field on the lungs." He agreed with the findings and conclusions of the prosector, Dr. Anderson.

Dr. Rizkalla was asked about the prosector's use of the phrase "consistent with." Dr. Bush had insisted that this phraseology indicated that the prosector's conclusion, in this case diagnosis, was equivocal. Dr. Rizkalla disagreed, and stated that "the word consistent carries the word of certainty." (*Id*. at 15). In this instance, he interpreted the description of the disease process and conclusions reached by the prosector, Dr. Anderson, as expressed with certainty.

The doctor explained his opinion as to the degree of pneumoconiosis on the basis of the amount of coal and silica dust and its distribution on the tissue. He agreed with the conclusions reached by other experts that the miner's interstitial pulmonary fibrosis was caused by coal dust exposure and coal workers' pneumoconiosis. In rendering his opinion as to a substantial burden of coal dust, Dr. Rizkalla looked to the clinical history, gross description of the lung, and microscopic slides.

Dr. Rizkalla detected macules or micronodules, and found anthracosilicosis. Dr. Rizkalla had no doubt as to the severity of the coal workers' pneumoconiosis present in this case. When asked about Dr. Bush's differentiation of discrete areas that were affected by pigment and those that were not, Dr. Rizkalla questioned how a pathologist could differentiate between a portion that was affected by coal dust and another portion that was not. He again opined that the fibrogenic agents present here would be the coal and silica dust.

When asked about Dr. Anderson's description of deposits of anthracotic pigment as "diffuse," Dr. Rizkalla said that the prosector found it in every field. The finding of "regional nodes" meant to Dr. Rizkalla that this was a reference to the lymphatic nodes that act as a filter. Dr. Rizkalla agreed that the "gold standard" for diagnosis would be the tissue examination. (*Id*.

at 23).

He testified that he found "abundant" silica, silicates and coal dust, and an "abundant" presence of mineral dust in the areas of fibrosis. He disagreed with Dr. Tomashefski's finding that the fibrosis was largely non-pigmented. (*Id*. at 25-26).

On cross-examination, Dr. Rizkalla agreed that interstitial pulmonary fibrosis and coal workers' pneumoconiosis represent two disease processes. He conceded that in his initial report he attributed the cause of death to interstitial pulmonary fibrosis. (*Id*. at 29-30). Dr. Rizkalla saw interstitial pulmonary fibrosis on a slide, but disagreed that there was no dust pigment. He disagreed with the others, including Dr. Perper, by saying that every slide had pigment and the pigment was evenly distributed.

Dr. Rizkalla explained:

> [The miner's simple coal workers' pneumoconiosis] is moderately severe. What is another entity, that there is diffuse interstitial fibrosis of the lung. Here's the question, if this diffuse interstitial fibrosis of the lung is induced by any etiology or there was no etiology here. So the etiology here was the substantial presence of the coal dust in the lung tissue induced this fibrotic process.
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> * * *
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> So you have here another response of the tissue. The response of these lungs, of that specific disease is responded by, and instead of localized fibrosis to create micro nodules, it's responded by diffuse interstitial pulmonary fibrosis and there are some pathologists and researchers who did a case study and published articles that certain people who had exposures to coal dust can respond by this interstitial pulmonary fibrosis, which histologically can mimic or have the same features as what's call[ed] idiopathic pulmonary fibrosis. But since we have an etiology, a cause, a reason, that we can identify in the slides that can induce fibrosis then we cannot ... consider it idiopathic pulmonary fibrosis.

(CX-8 at 33-34).

Dr. James R. Castle

Dr. Castle reviewed Mr. Plewa's medical records, including recent medical reports and deposition testimony, and submitted a detailed consultation report on November 17, 2003. (EX-3). He concluded that the miner suffered from coal workers' pneumoconiosis. Mr. Plewa certainly had sufficient coal mine dust exposure to cause pneumoconiosis, according to Dr.

Castle. The doctor also cited the miner's cigarette smoking history, which varied from 12 years at the rate of one pack per week to one-half a pack for 20 years, as sufficient to create an obstructive disease.

Dr. Castle offered an extensive explanation of idiopathic pulmonary fibrosis. Upon reviewing the entire medical course, he recounted that Mr. Plewa began exhibiting symptoms of breathlessness and cough between 1993 and 1995. He notes that "virtually all" the x-ray interpretations indicate the presence of interstitial pulmonary fibrosis at the lower lung zones.

He concluded that while the miner suffered from coal workers' pneumoconiosis, the primary pulmonary problem was idiopathic pulmonary fibrosis, a disease unrelated to coal mine dust exposure. Dr. Castle opined "with a reasonable degree of medical certainty based upon a review of all the data that [Mr. Plewa's] death was neither caused by, contributed to, [n]or hastened by the minimal, simple coal workers' pneumoconiosis found pathologically." He explained that the "physiologic studies that were done are absolutely classic for the presence of interstitial pulmonary fibrosis." He cited as well the absence of any obstructive component, noting that pneumoconiosis "generally" results in a mixed, irreversible obstructive and restrictive ventilatory impairment." Dr. Castle concluded that, while the miner also had some degree of centrilobular emphysema pathologically, "it was of insufficient severity to have caused any obstructive lung disease."

Dr. Castle is board certified in internal medicine and pulmonary diseases. He is also a B-reader. Dr. Castle presently is a Clinical Professor of Medicine at the University of Virginia College of Medicine, and has published in the area of diseases of unknown etiology. (EX-3).

Dr. Joseph F. Tomashefski

The deposition of Dr. Tomashefski was taken on December 11, 2003. (EX-6). He was asked to explicate his review of the medical records and files.

Dr. Tomashefski opined that the miner suffered from mild simple coal workers' pneumoconiosis (EX-6 at 39). He thought that the pneumoconiosis was so mild that it did not cause any clinical "manifestations or symptoms or impairment." He explained that the mild level of pneumoconiosis was confirmed by the absence of a diagnosis of pneumoconiosis on the x-rays. He concluded that the miner died as a result of respiratory failure as a result of progressive respiratory failure due to idiopathic pulmonary fibrosis. (*Id*. at 40-41).

He explained that the slides demonstrated a very mild simple coal workers' pneumoconiosis, and pointed out that there was diffuse interstitial fibrosis involving a majority of the slides; the fibrosis was long-standing, and had produced honeycomb changes under the pleura and was associated with a mild degree of inflammation. Dr. Tomashefski also said that in the affected areas there was "minimal black pigment and birefringent crystals." He also said that the miner's downhill course, which appeared to manifest itself in the records about six years before the miner's death, was additional confirmation of this disease. Dr. Tomashefski noted the x-ray and CT scan demonstrations of a "peripheral subpleural orientation of fibrosis, and honeycombing of the lung fields" as additional confirmation of idiopathic pulmonary fibrosis.

He then pointed out that the miner's clinical records excluded other classes of this pattern of fibrosis. (*Id.* at 43-44).

Dr. Tomashefski concluded that "there was no relationship between [Mr. Plewa's] exposure to coal mine dust or the presence of very mild simple coal workers' pneumoconiosis and the cause of his death." He also stated that the idiopathic pulmonary fibrosis was not derived from coal mine dust exposure. The fibrosis that afflicted the miner was also distinct from progressive massive fibrosis.

The doctor disagreed with Dr. Perper's characterization of this process as an interstitial fibrosis variant of coal workers' pneumoconiosis. Dr. Tomashefski explained that the pattern suggested by Dr. Perper does not appear in the literature. The doctor also disagreed with the opinion of Claimant's experts that one can never diagnose idiopathic pulmonary fibrosis. He acknowledged that coal workers can also develop IPF, but emphasized that, in this case, the degree of fibrosis is not commensurate with the minimal amount of pigment and birefringent crystals. He did not see the fibrous tissue impregnated with coal mine dust. Nor did Dr. Tomashefski detect that pattern of fibrosis that would be associated with coal mine dust or silica. (*Id*. at 49-52, 75).

On cross-examination, he explained "that the general distribution of pigment and birefringent crystals across all of the fibrous tissue was very minimal and it was an insufficient amount to explain this severe fibrosis." (*Id*. at 77). Dr. Tomashefski believed that the miner's pulmonary symptoms were manifest as early as 1993, although he acknowledged that symptoms could have commenced earlier. (*Id*. at 63).

Dr. Tomashefski testified that he did not credit the autopsy finding of emphysema. (*Id*. at 86). He said he did not address this disease because it didn't show up in the slides. Nor was there evidence of emphysema in the miner's clinical history, he said.

In response to Claimant's counsel's question concerning the cause of the miner's mild to moderate diffusion capacity in 1992, Dr. Tomashefski replied that he did not have a reference to that in his report. (*Id*. at 103). Employer's attorney reminded counsel that Dr. Perper had noted that this early condition had symptoms of sputum production and that it was treated by antibiotics — a suggestion that it was derived from an infection or inflammation.

Dr. Tomashefski was questioned about his familiarity with any literature that supported the proposition that "would indicate that coal dust exposure can cause a diffused interstitial fibrotic pattern that mimics or that may mimic idiopathic pulmonary fibrosis." He acknowledged that he was aware of such literature. (*Id*. at 118). Dr. Tomashefski did not agree that coal dust exposure can mimic IPF. In the end, Dr. Tomashefski agreed with the definition of IPF as "a progressive form of interstitial fibrosis of unknown etiology" with a "specific histologic pattern." (*Id*. at 146).

DISCUSSION

In assessing the probative value of the conflicting opinions, I must address the

explanations of experts' medical opinions, the documentation underlying their medical judgments, the sophistication and bases of their diagnoses, and their qualifications and credentials. *Sterling Smokeless Coal Co. v. Akers*, 131 F.3d 438, 441, 21 BLR 2-269 (4th Cir. 1997). *See also Clark v. Karst-Robbins Corp.*, 12 BLR 1-149 (1989)(*en banc*); *Lucostic v. United States Steel Corp.*, 8 BLR 1-46 (1985). The "mere statement of a physician, without any explanation of the basis for that statement, does not take the place of the required reasoning." *Lango v. Director, OWCP*, 104 F.3d 573, 577, 21 BLR 2-12 (3d Cir. 1997). *See also Director, OWCP v. Rowe*, 710 F.2d 251, 255 (6th Cir. 1983).

In this case, I do not fault the opinions of any expert for lack of explanation. The experts agree that Mr. Plewa died a respiratory death, and had suffered from an interstitial fibrosis. The point of contention is whether the miner's pneumoconiosis was involved in the development of his interstitial disease so that it hastened his death.

After review of the evidence of record, I find that Claimant has established that pneumoconiosis was a substantial contributing cause by hastening Mr. Plewa's death.[10] I credit the conclusions of Dr. Perper, as corroborated by Drs. Rizkalla and Schaaf and the conclusions from the autopsy, over the conflicting reports from Employer's experts, chiefly Drs. Bush, Castle, and Tomashefski.

I have carefully evaluated the views of Employer's experts, who deny that the miner suffered from more than a mild pneumoconiosis and that the effects of this disease would have had no impact on the course of his unrelated fibrosis. I note their emphatic disagreement with Claimant's experts as to the degree of coal workers' pneumoconiosis, or the extent of the residual effects of coal mine dust inhalation. I have also considered the view of Employer's experts that the nature of the fibrotic process that is exhibited in this case, as well as its primary locus and its architecture, all militate in their view against a finding of any involvement of pneumoconiosis in his death. I have accounted for their disagreement in theory with the view that pneumoconiosis would cause or contribute to the type of interstitial fibrosis process shown here. In addition, I have considered the fact, according to the medical history obtained by Dr. Smith in July, 2001, that Mr. Plewa had earlier been diagnosed with idiopathic interstitial fibrosis by a number of pulmonologists.

Initially, I accept the observations from Claimant's experts that the extent of the miner's coal workers' pneumoconiosis was greater than that found by Drs. Bush, Castle, and Tomashefski. In this regard, I note that the findings by the autopsy prosector, Dr. Anderson, who discovered "[d]ense deposits [of] anthracotic pigment with areas of scarring, hyalinization and scattered crystalline material," provide additional support for the observations by the

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[10] I am mindful that the miner's death certificate listed the immediate causes of death as "respiratory failure," and then "pulmonary fibrosis" as an underlying cause, as certified by Dr. Edward Scanlon. (DX-8). This conclusion does not significantly detract from the opinions that the miner suffered from coal workers' pneumoconiosis, and that the disease was a substantially contributing cause. *Cf. Smakula v. Weinberger*, 572 F.2d 127, 132 (3d Cir. 1978) (certificate's conclusion lacks support by the record).

pathologists who conducted a histological review.[11] The pigment was "identified diffusely." Because I find the opinions as to the extent of the miner's pneumoconiosis are credible, I likewise credit the corollary opinions that coal workers' pneumoconiosis was involved in the development of the miner's interstitial pulmonary fibrosis.

In addition, I find that the cross-examinations of both Dr. Tomashefski and Dr. Bush were exceptionally effective. Each physician was confronted with contrary findings of experts who found greater amounts of anthracotic pigment, which was opined to be an indicia of pneumoconiosis, and by evidence that the miner had exhibited pulmonary or respiratory symptoms to some extent well before his death. One basis, it appears, for positing the miner's death as due to IPF was the recognition that an average survival rate for persons afflicted with that disease would be approximately six years. Certainly, the fact that the miner may have been symptomatic to some extent outside the average survival span does not dictate that the miner was not afflicted with IPF. In this regard, I note the testimony of Mrs. Plewa, whom I consider a credible witness, who recalled that her husband exhibited symptoms as far back to 1990. This testimony, while not competent evidence to establish causation or etiology, is nonetheless probative as to the duration of the miner's symptoms. *See Soubic*, ___ F.3d at ___, n. 11, slip op. at 10 n.11.

Moreover, I find that the medical opinions of Employer's experts have not "persuasively discounted the effects" of Mr. Plewa's coal mine dust exposure in formulating their opinions that his interstitial disease, and death, was in no way associated with his coal mine employment. *See Peabody Coal Co. v. Hill*, 123 F.3d 412, 417, 21 BLR 1-192 (6th Cir. 1997). *See also*, *e.g.*, *Pastovich v. Consolidation Coal Co.*, BRB No. 98-0697 BLA (Feb. 11, 1999) (unpub.) (ALJ could properly criticize experts for failing to account persuasively for length of miner's coal mine dust exposure), *aff'd*, 216 F.3d 1075 (3d Cir. May 17, 2000) (table). In *Clinchfield Coal Co. v. Mullins*, 1999 WL 21329 (4th Cir. Jan. 20, 1999), the court concluded that the administrative law judge in that case permissibly discounted on two alternate grounds the medical opinions of Dr. Tomashefski, who had concluded in that case that the diseased miner's

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[11] Certainly, a prosector's opinion may not be credited on the basis of that expert's status where there is nothing in a record that suggests that access to the body enhanced the accuracy of the opinion. *See Bill Branch Coal Corp. v. Sparks*, 213 F.3d 186, 192 n. 6, 22 BLR 2-251 (4th Cir. 2000); *Urgolites v. BethEnergy Mines, Inc.*, 17 BLR 1-20 (1992). Moreover, a reviewing consultant may provide a persuasive opinion regarding the nature and extent of a miner's pneumoconiosis based on a histological review. *See generally Peabody Coal Co. v. McCandless*, 255 F.3d 465, 22 BLR 2-311 (7th Cir. 2001). In this instance, however, the autopsy findings as to the nature and extent of the anthracotic pigmentation have been evaluated in conjunction with the opinions of reviewing pathologists. Dr. Perper, for example, emphasized the importance of a review of gross findings as an important complement to a histological analysis. In certain circumstances, a fact-finder may credit the opinion of an autopsy prosector. *See Peabody Coal Co. v. Director*, *OWCP* [*Railey*], 972 F.2d 178, 182, 16 BLR 2-121 (7th Cir. 1992); *United States Steel Corp. v. Oravetz*, 686 F.2d 197, 200, 4 BLR 2-130 (3d Cir. 1982). Again, this is not a case of an automatic deference to the autopsy prosector, but a recognition of the relevance to, and support of, Dr. Anderson's findings on autopsy, for the medical opinions of Claimant's experts, especially with respect to the extent of anthracotic pigment.

fibrosis was idiopathic. The court approved the administrative law judge's explanation that the doctor "did not assess how [the miner's] thirty-six year exposure to coal dust related to the fibrosis that led to his death." *Mullins*, 1999 WL 21329 at **3.[12]

I note that Dr. Perper and others have referenced a coal mine employment history of 30 years or more. The parties have stipulated to 27 years. I have duly noted this discrepancy, and find that it does not significantly detract from the weight of those medical reports that find that pneumoconiosis hastened the miner's death. The length of time Mr. Plewa was in the mines was deemed by Dr. Bush, for example, to be of a sufficient duration of exposure to cause pneumoconiosis. Of considerable significance is Dr. Perper's opinion that the condition of the peri-bronchial lymph nodes was indicative of severe exposure consistent with occupational exposure of more than 30 years. (CX-6 [Deposition] at 24).

I have also carefully evaluated the credentials of the medical experts in this case. Both sides proffered the opinions of highly qualified physicians, most of whom have academic teaching experience as well. I note that Dr. Tomashefski is a full professor in his field, and has extensive experience with occupational diseases, if not coal workers' pneumoconiosis. *See Worhach v. Director*, *OWCP*, 17 BLR 1-105 (1993). I also note, however, that Drs. Perper and Rizkalla have considerable experience in conducting post-mortem examinations on decedent miners.

In the final analysis, I am persuaded by the conclusions of Claimant's experts that the miner's coal workers' pneumoconiosis was a substantial contributing factor because it hastened his death.

ATTORNEY FEE

No award of attorney's fees for services rendered to Claimant is made herein since no application has been received. Thirty days is hereby allowed to Claimant's counsel for the submission of such an application. In its preparation, counsel's attention is directed to 20 C.F.R. §§ 725.365 and 366. A service sheet showing that service has been made upon all parties, including Claimant, must accompany the application. Parties have ten (10) days following receipt of the application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

ORDER

The claim of ROSE M. PLEWA for survivor's benefits under the Act is hereby GRANTED.

Accordingly, BETHENERGY MINES, INC. shall:

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[12] These unpublished decisions are not cited for precedential authority, but are illustrative of the rationale utilized by administrative law judges who have discounted medical opinions that do not adequately account for the miner's lengthy coal mine dust exposure.

1. Pay ROSE M. PLEWA benefits on her survivor's claim, with the onset of benefits commencing January 1, 2002.

2. Pay Claimant's attorney, Robert J. Bilonick, Esquire, fees and expenses to be established in a Supplemental Decision and Order.

A

STEPHEN L. PURCELL
Administrative Law Judge

Washington, D.C.

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within thirty (30) days from the date of this Decision and Order by filing a Notice of Appeal with the Benefits Review Board at P.O. Box 37601, Washington, D.C. 20013-7601. A copy of this notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, Room N-2117, 200 Constitution Avenue, N.W., Washington, D.C. 20210.